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\* \* \* \* \* STN Columbus \* \* \* \* \*

FILE 'HOME' ENTERED AT 11:21:24 ON 01 AUG 2001

=> file medline biosis embase caplus uspatfull

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

0.21

0.21

FILE 'MEDLINE' ENTERED AT 11:21:36 ON 01 AUG 2001

FILE 'BIOSIS' ENTERED AT 11:21:36 ON 01 AUG 2001

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FILE 'EMBASE' ENTERED AT 11:21:36 ON 01 AUG 2001

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FILE 'CAPLUS' ENTERED AT 11:21:36 ON 01 AUG 2001

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FILE 'USPATFULL' ENTERED AT 11:21:36 ON 01 AUG 2001

CA INDEXING COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY (ACS)

=> s potassium (p) channel (p) interact? (p) protein

L1 1054 POTASSIUM (P) CHANNEL (P) INTERACT? (P) PROTEIN

=> s potassium (p) channel (p) interact? (p) protein (p) congestive

4 FILES SEARCHED...

L2 0 POTASSIUM (P) CHANNEL (P) INTERACT? (P) PROTEIN (P) CONGESTIVE

=> s potassium (p) channel (p) interact? (p) protein (p) long

4 FILES SEARCHED...

L3 50 POTASSIUM (P) CHANNEL (P) INTERACT? (P) PROTEIN (P) LONG

=> s potassium (p) channel (p) interact? (p) protein (p) long (p) qt

L4 20 POTASSIUM (P) CHANNEL (P) INTERACT? (P) PROTEIN (P) LONG (P) QT

=> dup rem l4

PROCESSING COMPLETED FOR L4

L5 8 DUP REM L4 (12 DUPLICATES REMOVED)

=> s potassium (a) channel (a) interact? (a) protein

L6 15 POTASSIUM (A) CHANNEL (A) INTERACT? (A) PROTEIN

=> dup rem l6

PROCESSING COMPLETED FOR L6

L7 12 DUP REM L6 (3 DUPLICATES REMOVED)

=> d his

(FILE 'HOME' ENTERED AT 11:21:24 ON 01 AUG 2001)

FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS, USPATFULL' ENTERED AT 11:21:36 ON 01 AUG 2001

L1 1054 S POTASSIUM (P) CHANNEL (P) INTERACT? (P) PROTEIN  
L2 0 S POTASSIUM (P) CHANNEL (P) INTERACT? (P) PROTEIN (P)  
CONGESTIV  
L3 50 S POTASSIUM (P) CHANNEL (P) INTERACT? (P) PROTEIN (P) LONG  
L4 20 S POTASSIUM (P) CHANNEL (P) INTERACT? (P) PROTEIN (P) LONG  
(P)  
L5 8 DUP REM L4 (12 DUPLICATES REMOVED)  
L6 15 S POTASSIUM (A) CHANNEL (A) INTERACT? (A) PROTEIN  
L7 12 DUP REM L6 (3 DUPLICATES REMOVED)

=> d 15 total ibib kwic

L5 ANSWER 1 OF 8 MEDLINE DUPLICATE 1  
ACCESSION NUMBER: 1998215377 MEDLINE  
DOCUMENT NUMBER: 98215377 PubMed ID: 9556090  
TITLE: Genetics, molecular mechanisms and management of long QT syndrome.  
AUTHOR: Wang Q; Chen Q; Towbin J A  
CORPORATE SOURCE: Department of Pediatrics, Baylor College of Medicine, Texas  
Children's Hospital, Houston 77030, USA..  
qwang@bcm.tmc.edu  
SOURCE: ANNALS OF MEDICINE, (1998 Feb) 30 (1) 58-65. Ref: 63  
Journal code: AMD; 8906388. ISSN: 0785-3890.  
PUB. COUNTRY: ENGLAND: United Kingdom  
Journal; Article; (JOURNAL ARTICLE)  
General Review; (REVIEW)  
(REVIEW, TUTORIAL)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 199806  
ENTRY DATE: Entered STN: 19980618  
Last Updated on STN: 19980618  
Entered Medline: 19980611  
AB Cardiac arrhythmias cause more than 300,000 sudden deaths each year in the  
USA alone. **Long QT** syndrome (LQT) is a cardiac disorder that causes sudden death from ventricular tachyarrhythmias, specifically torsade de pointes. Four LQT genes. . . (LQT2) on chromosome 7q35-36, SCN5A (LQT3) on chromosome 3p21-24, and MinK (LQT5) on chromosome 21q22. SCN5A encodes the cardiac sodium **channel**, and LQT-causing mutations in SCN5A lead to the generation of a late phase of inactivation-resistant whole-cell inward currents. Mexiletine, a sodium **channel** blocker, is effective in shortening the **QT** interval corrected for heart rate (QTc) of patients with SCN5A mutations. HERG encodes the cardiac I(Kr) **potassium channel**. Mutations in HERG act by a dominant-negative mechanism or by a loss-of-function mechanism. Raising the serum **potassium** concentration can increase outward HERG **potassium** current and is effective in shortening the QTc of patients with HERG mutations. KVLQT1 is a cardiac **potassium channel protein** that **interacts** with another small **potassium channel** MinK to form the cardiac I(Ks) **potassium channel**. Like HERG mutations, mutations in KVLQT1 and MinK can act by a dominant-negative mechanism or a loss-of-function mechanism. An effective.

. . . LQT patients with KVLQT1 or MinK mutations is expected to be developed based on the functional characterization of the I(Ks) **potassium channel**. Genetic testing is now available for some patients with LQT.

L5 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 1997:506691 CAPLUS  
DOCUMENT NUMBER: 127:118273  
TITLE: KVLQT1 is a long QT syndrome gene whose protein product coassembles with minK to form cardiac IKs potassium channels  
INVENTOR(S): Keating, Mark F.; Curran, Mark E.; Landes, Gregory M.; Connors, Timothy D.  
PATENT ASSIGNEE(S): University of Utah Research Foundation, USA; Genzyme Genetics  
SOURCE: PCT Int. Appl., 116 pp.  
CODEN: PIXXD2  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 2  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9723632	A1	19970703	WO 1996-US19917	19961220
W: AU, CA, JP, KR, NZ				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9719512	A1	19970717	AU 1997-19512	19961220
AU 714041	B2	19991216		
EP 87649	A1	19981111	EP 1996-946233	19961220
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2001501445	T2	20010206	JP 1997-523726	19961220
PRIORITY APPLN. INFO.:			US 1995-19014	P 19951222
			US 1996-739383	A 19961029
			WO 1996-US19917	W 19961220

AB One aspect of the invention relates to the identification of the mol. basis of **long QT** syndrome. KVLQT1 cDNA was isolated from a human heart cDNA library, and its 581-amino-acid **protein** product was structurally and functionally characterized; a Xenopus KVLQT1 homolog was also identified. More specifically, mutated KVLQT1 is shown to cause **long QT** syndrome. Anal. of this gene will provide an early diagnosis of subjects with **long QT** syndrome. The diagnostic methods comprise analyzing the nucleic acid sequence of the KVLQT1 gene of an individual to be tested and comparing them with the nucleic acid sequence of the native, non-variant gene. Alternatively, the amino acid sequence of KVLQT1 may be analyzed for mutations which cause **long QT** syndrome. Presymptomatic diagnosis of **long QT** syndrome will enable practitioners to treat this disorder using existing medical therapy. A second aspect of the invention relates to the realization that KVLQT1 coassemble with minK to form a cardiac **potassium channel**. This allows one to assay for drugs which **interact** with this **channel** to identify new drug which are useful for treating or preventing **long QT**.

L5 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 1997:506918 CAPLUS  
DOCUMENT NUMBER: 127:118275  
TITLE: KVLQT1 is a long QT syndrome gene whose protein product coassembles with minK to form cardiac IKs potassium channels  
INVENTOR(S): Keating, Mark T.; Sanguinetti, Michael C.; Curran,

Mark E.  
PATENT ASSIGNEE(S): University of Utah Research Foundation, USA  
SOURCE: PCT Int. Appl., 105 pp.  
CODEN: PIXXD2  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 2  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9723598	A2	19970703	WO 1996-US19756	19961220
W: AU, CA, JP, KR, NZ				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9717428	A1	19970717	AU 1997-17428	19961220
AU 714527	B2	20000106		
EP 870041	A2	19981014	EP 1996-945935	19961220
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.:  
US 1995-19014 P 19951222  
US 1996-739383 A 19961029  
WO 1996-US19756 W 19961220

AB One aspect of the invention relates to the identification of the mol. basis of **long QT** syndrome. KVLQT1 cDNA was isolated from a human heart cDNA library, and its 581-amino-acid **protein** product was structurally and functionally characterized; a Xenopus KVLQT1 homolog was also identified. Mutations in KVLQT1 are shown to cosegregate with the **long QT** syndrome. More specifically, minK is found to coassemble with KVQLT1 to form a cardiac **potassium channel**. Thus, mutated minK causes **long QT** syndrome. Anal. of this gene will provide an early diagnosis of subjects with **long QT** syndrome. The diagnostic methods comprise analyzing the nucleic acid sequence of the minK gene of an individual to be tested and comparing them with the nucleic acid sequence of the native, non-variant gene. Alternatively, the amino acid sequence of minK may be analyzed for mutations which cause **long QT** syndrome. Presymptomatic diagnosis of **long QT** syndrome will enable practitioners to treat this disorder using existing medical therapy. Assays are also provided for drugs which **interact** with the cardiac **potassium channel** to identify new drugs which are useful for treating or preventing **long QT**.

L5 ANSWER 4 OF 8 MEDLINE DUPLICATE 2  
ACCESSION NUMBER: 97472471 MEDLINE  
DOCUMENT NUMBER: 97472471 PubMed ID: 9328483  
TITLE: Isk and KvLQT1: mutation in either of the two subunits of the slow component of the delayed rectifier potassium channel can cause Jervell and Lange-Nielsen syndrome.  
AUTHOR: Tyson J; Tranebjaerg L; Bellman S; Wren C; Taylor J F; Bathen J; Aslaksen B; Sorland S J; Lund O; Malcolm S; Pembrey M; Bhattacharya S; Bitner-Glindzicz M  
CORPORATE SOURCE: Unit of Clinical Genetics, Institute of Child Health and Great Ormond Street Hospital for Children NHS Trust, UCL Medical School, 30 Guilford Street, London WC1N 1EH, UK.  
SOURCE: HUMAN MOLECULAR GENETICS, (1997 Nov) 6 (12) 2179-85.  
Journal code: BRC; 9208958. ISSN: 0964-6906.  
PUB. COUNTRY: ENGLAND: United Kingdom  
Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 199712  
ENTRY DATE: Entered STN: 19980109  
Last Updated on STN: 19990129

Entered Medline: 19971219

AB . . . sensorineural deafness associated with syncopal episodes. These are caused by ventricular arrhythmias secondary to abnormal repolarisation, manifested by a prolonged **QT** interval on the electrocardiogram. Recently, in families with JLNS, Neyroud et al. reported homozygosity for a single mutation in KVLQT1, a gene which has previously been shown to be mutated in families with dominantly inherited isolated **long QT** syndrome [Neyroud et al. (1997) Nature Genet., 15, 186-189]. We have analysed a group of families with JLNS. . . by descent for markers on chromosome 21, in a region containing the gene IsK. This codes for a transmembrane **protein** known to associate with KVLQT1 to form the slow component of the delayed rectifier **potassium channel**. Sequencing of the affected boys showed a homozygous mutation, demonstrating that mutation in the IsK gene may be a rare cause of JLNS and that an indistinguishable phenotype can arise from mutations in either of the two **interacting** molecules.

L5 ANSWER 5 OF 8 MEDLINE DUPLICATE 3  
ACCESSION NUMBER: 97462827 MEDLINE  
DOCUMENT NUMBER: 97462827 PubMed ID: 9323054  
TITLE: Dominant-negative KvLQT1 mutations underlie the LQT1 form of long QT syndrome.  
COMMENT: Comment in: Circulation. 1997 Sep 16;96(6):1720-1  
AUTHOR: Shalaby F Y; Levesque P C; Yang W P; Little W A; Conder M L; Jenkins-West T; Blannar M A  
CORPORATE SOURCE: Department of Cardiovascular Drug Discovery, Bristol-Myers Squibb Pharmaceutical Research Institute, Princeton, NJ 08543-4000, USA.  
SOURCE: CIRCULATION, (1997 Sep 16) 96 (6) 1733-6.  
PUB. COUNTRY: Journal code: DAW; 0147763. ISSN: 0009-7322.  
LANGUAGE: English  
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals  
ENTRY MONTH: 199710  
ENTRY DATE: Entered STN: 19971105  
Last Updated on STN: 19990129  
Entered Medline: 19971022

AB BACKGROUND: Mutations that map to the KvLQT1 gene on human chromosome 11 account for more than 50% of inherited **long QT** syndrome (LQTS). It has been discovered recently that the KvLQT1 and mink **proteins** functionally **interact** to generate a current with biophysical properties similar to I(Ks), the slowly activating delayed-rectifier cardiac **potassium** current. Since I(Ks) modulates the repolarization of cardiac action potentials it is reasonable to hypothesize that mutations in KvLQT1 reduce. . . S2-S3 cytoplasmic loop (A177P) or threonine with isoleucine in the highly conserved signature sequence of the pore (T311I) yield inactive **channels** when expressed individually, whereas substitution of leucine with phenylalanine in the S5 transmembrane domain (L272F) yields a functional **channel** with reduced macroscopic conductance. However, all these mutants inhibit wild-type KvLQT1 currents in a dominant-negative fashion.  
CONCLUSIONS: In LQTS-affected individuals. . .

L5 ANSWER 6 OF 8 MEDLINE DUPLICATE 4  
ACCESSION NUMBER: 97150816 MEDLINE  
DOCUMENT NUMBER: 97150816 PubMed ID: 8995352  
TITLE: The human delta1261 mutation of the HERG potassium channel results in a truncated protein that contains a subunit interaction domain and decreases the channel expression.  
AUTHOR: Li X; Xu J; Li M  
CORPORATE SOURCE: Department of Physiology, The Johns Hopkins University School of Medicine, Baltimore, Maryland 21205, USA.

SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1997 Jan 10) 272 (2) 705-8.  
Journal code: HIV; 2985121R. ISSN: 0021-9258.  
PUB. COUNTRY: United States  
Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 199702  
ENTRY DATE: Entered STN: 19970227  
Last Updated on STN: 19970227  
Entered Medline: 19970212

AB HERG (human eag-related gene) encodes an inward-rectifier **potassium channel** formed by the assembly of four subunits. Since the truncated HERG **protein** in patients with **long QT** syndrome induces a dominant phenotype, that is, cardiac sudden death, the assembly of nonfunctional complexes between wild-type and mutated subunits. . . . To understand HERG-mediated cardiac sudden death at the molecular level, it is important to determine which regions in the HERG **protein** participate in subunit **interaction**. We therefore report the identification of a subunit **interaction** domain, NAB(HERG), that is localized at the hydrophilic cytoplasmic N terminus and can form a tetramer in the absence of the rest of the HERG **protein**. Truncated HERG **proteins** containing NAB(HERG), including one that resulted from the delta1261 human mutation, inhibit the functional expression of the HERG **channel** in transfected cells. Together, these results support the notion that the expression of HERG in the human heart may be decreased in the presence of the truncated subunit. Such a decrease of **potassium channel** expression can contribute to the longer **QT** intervals observed in the patients with the HERG mutation.

L5 ANSWER 7 OF 8 BIOSIS COPYRIGHT 2001 BIOSIS  
ACCESSION NUMBER: 1997:175428 BIOSIS  
DOCUMENT NUMBER: PREV199799467141  
TITLE: Building (potassium) channels to the 21st century  
(Neuropharmacology IV: K<sup>+</sup> channels: Washington, D.C., USA, November 14-16, 1996.  
AUTHOR(S): Trimmer, James S. (1); Rhodes, Kenneth J.  
CORPORATE SOURCE: (1) Dep. Biochemistry Cell Biology, Inst. Cell Dev.  
Biology, State Univ. New York Stony Brook, Stony Brook, NY 11794 USA  
SOURCE: Trends in Neurosciences, (1997) Vol. 20, No. 3, pp. 99-100.  
ISSN: 0166-2236.  
DOCUMENT TYPE: Conference; Report  
LANGUAGE: English  
IT Miscellaneous Descriptors  
BIOCHEMISTRY AND BIOPHYSICS; GENOME SEQUENCE; HEART DISEASE;  
**LONG QT SYNDROME; POTASSIUM CHANNELS; PROTEIN-PROTEIN INTERACTIONS; RESTING MEMBRANE POTENTIAL; SHAKER PROTEIN**

L5 ANSWER 8 OF 8 MEDLINE  
ACCESSION NUMBER: 95406767 MEDLINE  
DOCUMENT NUMBER: 95406767 PubMed ID: 7676324  
TITLE: Molecular genetics of long QT syndrome.  
AUTHOR: Keating M T  
CORPORATE SOURCE: Department of Human Genetics, University of Utah, Salt Lake City 84112, USA.  
CONTRACT NUMBER: R01HL48074 (NHLBI)  
SOURCE: SOCIETY OF GENERAL PHYSIOLOGISTS SERIES, (1995) 50 53-60.  
Ref: 28

PUB. COUNTRY: Journal code: UU2; 0433431. ISSN: 0094-7733.  
United States  
Journal; Article; (JOURNAL ARTICLE)  
General Review; (REVIEW)  
(REVIEW, TUTORIAL)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 199510  
ENTRY DATE: Entered STN: 19951026  
Last Updated on STN: 19951026  
Entered Medline: 19951019

AB In the **long QT** syndrome (LQT), individuals suffer from syncope, seizures and sudden death due to cardiac arrhythmias, specifically torsade de pointes and ventricular fibrillation. Many of these individuals also have prolongation of the **QT** interval on electrocardiograms, suggesting abnormal cardiac repolarization. To improve our understanding of the mechanisms underlying LQT and to facilitate presymptomatic. . . making this protoncogene a candidate for LQT. This hypothesis was supported by physiologic data; other investigators had shown that ras **proteins** modulate cardiac **potassium channels** and an abnormality of **potassium** homeostasis could explain LQT. We eliminated HRAS as a candidate, however, by sequencing the coding region in 10 unrelated patients. . . unlinked, at least one more LQT locus exists. This degree of heterogeneity presents opportunities. It seems likely, for example, that **proteins** encoded by distinct LQT genes **interact** to modulate cardiac repolarization. (ABSTRACT TRUNCATED AT 250 WORDS)

=> d 17 total ibib kwic

L7 ANSWER 1 OF 12 MEDLINE DUPLICATE 1  
ACCESSION NUMBER: 2001336578 MEDLINE  
DOCUMENT NUMBER: 21297487 PubMed ID: 11404400  
TITLE: Kinetic modulation of Kv4-mediated A-current by arachidonic acid is dependent on **potassium channel interacting proteins**.  
AUTHOR: Holmqvist M H; Cao J; Knoppers M H; Jurman M E; Distefano P  
CORPORATE SOURCE: S; Rhodes K J; Xie Y; An W F  
Millennium Pharmaceuticals Inc., Cambridge, Massachusetts 02139, USA.  
SOURCE: JOURNAL OF NEUROSCIENCE, (2001 Jun 15) 21 (12) 4154-61.  
Journal code: JDF; 8102140. ISSN: 1529-2401.  
PUB. COUNTRY: United States  
Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 200107  
ENTRY DATE: Entered STN: 20010716  
Last Updated on STN: 20010716  
Entered Medline: 20010712  
TI Kinetic modulation of Kv4-mediated A-current by arachidonic acid is dependent on **potassium channel interacting proteins**.

L7 ANSWER 2 OF 12 BIOSIS COPYRIGHT 2001 BIOSIS  
ACCESSION NUMBER: 2001:199211 BIOSIS  
DOCUMENT NUMBER: PREV200100199211  
TITLE: K-Channel Interacting Protein dependent modulation of Kv4 current by fatty acids.  
AUTHOR(S): Holmqvist, Mats H. (1); Cao, Jie (1); Jurman, Mark E. (1);

Xie, Yu (1); Rhodes, Kenneth J.; Distefano, Peter S. (1);  
An, W. Frank (1)

CORPORATE SOURCE: (1) Millennium Pharmaceuticals, Inc., Cambridge, MA, 02139  
USA

SOURCE: Biophysical Journal, (January, 2001) Vol. 80, No. 1 Part  
2,  
pp. 506a. print.  
Meeting Info.: 45th Annual Meeting of the Biophysical  
Society Boston, Massachusetts, USA February 17-21, 2001  
Biophysical Society  
. ISSN: 0006-3495.

DOCUMENT TYPE: Conference  
LANGUAGE: English  
SUMMARY LANGUAGE: English

IT . . .  
neuron: dendritic A current, nervous system

IT Chemicals & Biochemicals  
Kv4 alpha-subunit; Kv4 potassium channel: modulation; arachidonic  
acid;  
fatty acids; **potassium channel-interacting  
protein**

L7 ANSWER 3 OF 12 BIOSIS COPYRIGHT 2001 BIOSIS  
ACCESSION NUMBER: 2001:173636 BIOSIS  
DOCUMENT NUMBER: PREV200100173636  
TITLE: Kv4 channel gating and effects of the K channel  
interacting  
proteins (KChIPs).

AUTHOR(S): Baehring, Robert (1); Gebauer, Manuel (1); Boland, Linda  
M.  
(1); Pongs, Olaf (1); Isbrandt, Dirk (1)

CORPORATE SOURCE: (1) Institut fuer Neurale Signalverarbeitung, Martinistr.  
52, 20246, Hamburg Germany

SOURCE: Biophysical Journal, (January, 2001) Vol. 80, No. 1 Part  
2,  
pp. 439a. print.  
Meeting Info.: 45th Annual Meeting of the Biophysical  
Society Boston, Massachusetts, USA February 17-21, 2001  
Biophysical Society  
. ISSN: 0006-3495.

DOCUMENT TYPE: Conference  
LANGUAGE: English  
SUMMARY LANGUAGE: English

IT Major Concepts  
Biochemistry and Molecular Biophysics; Cell Biology

IT Chemicals & Biochemicals  
Kv4 channel: gating; **potassium channel  
interaction protein** [KCHIPs]

L7 ANSWER 4 OF 12 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.  
ACCESSION NUMBER: 2001219091 EMBASE  
TITLE: Different effects of the Ca(2+)-binding protein, KChIP1,  
on  
two Kv4 subfamily members, Kv4.1 and Kv4.2.

AUTHOR: Nakamura T.Y.; Nandi S.; Pountney D.J.; Artman M.; Rudy  
B.;  
Coetzee W.A.

CORPORATE SOURCE: T.Y. Nakamura, Pediatric Cardiology (TH519), NYU School of  
Medicine, 560 First Avenue, New York, NY 10016, United  
States. tomoe.nishitani@med.nyu.edu

SOURCE: FEBS Letters, (22 Jun 2001) 499/3 (205-209).  
Refs: 27  
ISSN: 0014-5793 CODEN: FEBLAL

PUBLISHER IDENT.: S 0014-5793(01)02560-1  
COUNTRY: Netherlands  
DOCUMENT TYPE: Journal; Article



FILE SEGMENT: 029 Clinical Biochemistry  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
CT Medical Descriptors:  
\*protein . . . interaction  
protein family  
potassium channel  
potassium current  
chimera  
amino terminal sequence  
biodiversity  
protein expression  
nonhuman  
controlled study  
animal cell  
article  
nucleotide sequence  
priority journal  
\*calcium binding protein: EC, endogenous compound  
\*protein kv4: EC, endogenous compound  
**\*potassium channel interacting protein 1: EC, endogenous compound**  
\*protein subunit: EC, endogenous compound  
unclassified drug

L7 ANSWER 5 OF 12 BIOSIS COPYRIGHT 2001 BIOSIS  
ACCESSION NUMBER: 2001:208654 BIOSIS  
DOCUMENT NUMBER: PREV200100208654  
TITLE: Molecular cloning and expression of the novel splice  
variants of K<sup>+</sup> channel-interacting protein 2.  
AUTHOR(S): Ohya, Susumu; Morohashi, Yuichi; Muraki, Katsuhiko;  
Tomita, Taisuke; Watanabe, Minoru; Iwatsubo, Takeshi; Imaizumi,  
Yuji (1)  
CORPORATE SOURCE: (1) Department of Molecular and Cellular Pharmacology,  
Faculty of Pharmaceutical Sciences, Nagoya City  
University,  
3-1 Tanabedori, Mizuhoku, Nagoya, 467-8603:  
yimaizum@phar.nagoya-cu.ac.jp Japan  
SOURCE: Biochemical and Biophysical Research Communications,  
(March 23, 2001) Vol. 282, No. 1, pp. 96-102. print.  
ISSN: 0006-291X.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
IT . . .  
Organisms  
aorta: circulatory system; brain: nervous system; heart: circulatory  
system; kidney: excretory system  
IT Chemicals & Biochemicals  
cDNA [complementary DNA]; **potassium channel-**  
**interacting protein 2** [KChIP2]: expression, molecular  
cloning

L7 ANSWER 6 OF 12 CAPLUS COPYRIGHT 2001 ACS  
ACCESSION NUMBER: 2000:368431 CAPLUS  
DOCUMENT NUMBER: 133:13914  
TITLE: Proteins interacting with potassium channel proteins  
identified in two-hybrid systems and datamining  
INVENTOR(S): Rhodes, Kenneth; Betty, Maria; Ling, Huai-ping; An,  
Wengian  
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., USA; American Home  
Products Corporation  
SOURCE: PCT Int. Appl., 306 pp.  
CODEN: PIXXD2  
DOCUMENT TYPE: Patent

LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 1  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000031133	A2	20000602	WO 1999-US27428	19991119
WO 2000031133	A3	20001005		

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ

RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:

US 1998-109333	P	19981120
US 1998-110033	P	19981125
US 1998-110277	P	19981130
US 1999-298731	A2	19990423
US 1999-350614	A2	19990709
US 1999-350874	A2	19990709
US 1999-399913	A2	19990921
US 1999-400492	A2	19990921

ST **potassium channel interacting protein** gene cloning; mouse monkey **potassium channel interacting protein**; human rat **potassium channel interacting protein**; calcium binding **potassium channel interacting protein**

IT Proteins, specific or class  
 RL: BOC (Biological occurrence); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)  
 (PCIP (**potassium-channel interacting proteins**); proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Nervous system  
 (central, disease, effectors of **potassium channel-interacting protein** for treatment of; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Brain  
 (cerebellum, granular layer, **potassium-channel interacting proteins** mRNA in; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Brain  
 (corpus striatum, **potassium-channel interacting proteins** mRNA in; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Brain  
 (cortex, **potassium-channel interacting proteins** mRNA in; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Cardiovascular system  
 (disease, effectors of **potassium channel-interacting protein** for treatment of; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Anticonvulsants  
 (effectors of **potassium channel-interacting protein** as; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Epilepsy  
 (effectors of **potassium channel-interacting**

protein for treatment of; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Nucleic acid amplification (method)  
Nucleic acid hybridization  
(for detection of **potassium channel-interacting protein** gene expression; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Primers (nucleic acid)  
Probes (nucleic acid)  
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)  
(for detection of **potassium channel-interacting protein** gene expression; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Immunoassay  
(for detection of **potassium channel-interacting proteins**; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Susceptibility (genetic)  
(for disease assocd. with **potassium channel-interacting protein**, screening for; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Drug screening  
(for effectors of **potassium channel-interacting protein**; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Gene, animal  
RL: BOC (Biological occurrence); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)  
(for **potassium channel interacting proteins**; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT cDNA sequences  
(for **potassium channel-interacting proteins** of human, mouse, rat, and monkey; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT mRNA  
RL: BOC (Biological occurrence); BIOL (Biological study); OCCU (Occurrence)  
(for **potassium-channel interacting proteins**, tissue distribution of; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Brain  
(habenula, medial nucleus, **potassium-channel interacting proteins** mRNA in; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Brain  
(hippocampus, **potassium-channel interacting proteins** mRNA in; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Chromosome  
(human 10, 10q22-q24, **potassium-channel interacting protein** gene mapping to; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT EF hand  
(in **potassium-channel interacting proteins**; proteins interacting with potassium channel proteins identified in two-hybrid systems and datamining)

IT Brain  
 (midbrain, **potassium-channel interacting**  
**proteins** mRNA in; proteins interacting with potassium channel  
 proteins identified in two-hybrid systems and datamining)

IT Diagnosis  
 (mol., of disease assocd. with **potassium channel-**  
**interacting protein**; proteins interacting with  
 potassium channel proteins identified in two-hybrid systems and  
 datamining)

IT Molecular cloning  
 (of cDNAs for **potassium channel-interacting**  
**proteins** of human, mouse, rat, and monkey; proteins interacting  
 with potassium channel proteins identified in two-hybrid systems and  
 datamining)

IT Protein sequences  
 (of **potassium channel-interacting**  
**proteins** of human, mouse, rat, and monkey; proteins interacting  
 with potassium channel proteins identified in two-hybrid systems and  
 datamining)

IT Genetic mapping  
 (of **potassium-channel interacting**  
**protein** gene of human; proteins interacting with potassium  
 channel proteins identified in two-hybrid systems and datamining)

IT Calcium-binding **proteins**  
 RL: BSU (Biological study, unclassified); BIOL (Biological study)  
 (**potassium-channel interacting**  
**proteins** as; proteins interacting with potassium channel  
 proteins identified in two-hybrid systems and datamining)

IT Brain  
 Heart  
 (**potassium-channel interacting**  
**proteins** mRNA in; proteins interacting with potassium channel  
 proteins identified in two-hybrid systems and datamining)

IT Nervous system  
 (spinocerebellar ataxia, effectors of **potassium**  
**channel-interacting protein** for treatment  
 of; proteins interacting with potassium channel proteins identified in  
 two-hybrid systems and datamining)

IT Brain  
 (stem, **potassium-channel interacting**  
**proteins** mRNA in; proteins interacting with potassium channel  
 proteins identified in two-hybrid systems and datamining)

IT Brain  
 (substantia nigra, **potassium-channel**  
**interacting proteins** mRNA in; proteins interacting  
 with potassium channel proteins identified in two-hybrid systems and  
 datamining)

IT Brain  
 (superior colliculus, **potassium-channel**  
**interacting proteins** mRNA in; proteins interacting  
 with potassium channel proteins identified in two-hybrid systems and  
 datamining)

IT Brain  
 (thalamus, **potassium-channel interacting**  
**proteins** mRNA in; proteins interacting with potassium channel  
 proteins identified in two-hybrid systems and datamining)

IT Antibodies  
 RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)  
 (to **potassium channel-interacting**  
**proteins**; proteins interacting with potassium channel proteins  
 identified in two-hybrid systems and datamining)

L7 ANSWER 7 OF 12 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: 2001:101622 BIOSIS

DOCUMENT NUMBER: PREV200100101622

TITLE: Kinetic modulation of Kv4-current by arachidonic acid is

dependent on K-channel interacting proteins.

AUTHOR(S): Holmqvist, M. H. (1); Xie, Y.; Jurman, M. E.; Cao, J.; Rhodes, K. J.; Distefano, P. S.; An, W. F.

CORPORATE SOURCE: (1) Millennium Pharmaceuticals, Inc., Cambridge, MA USA

SOURCE: Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-614.3. print.  
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience New Orleans, LA, USA November 04-09, 2000  
Society for Neuroscience  
. ISSN: 0190-5295.

DOCUMENT TYPE: Conference

LANGUAGE: English

SUMMARY LANGUAGE: English

IT . . . . .  
Biochemistry and Molecular Biophysics

IT Parts, Structures, & Systems of Organisms  
oocytes: reproductive system

IT Chemicals & Biochemicals  
arachidonic acid; **potassium-channel  
interacting proteins**; voltage-gated fast-activating  
potassium channels: inactivation

L7 ANSWER 8 OF 12 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: 2001:102702 BIOSIS

DOCUMENT NUMBER: PREV200100102702

TITLE: K-channel interacting protein-2 splice variants,  
chromosomal organization and localization.

AUTHOR(S): An, W. F. (1); Ling, H. P.; Chen, H.; Xie, Y.; Holmqvist, M. H.; Cao, J.; Jurman, M. E.; Dussault, B. J.; Distefano, P. S.; Betty, M.; Mendoza, G.; Bowlby, M. R.; Rhodes, K. J.

CORPORATE SOURCE: (1) Millennium Pharmaceuticals, Cambridge, MA USA

SOURCE: Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-614.2. print.  
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience New Orleans, LA, USA November 04-09, 2000  
Society for Neuroscience  
. ISSN: 0190-5295.

DOCUMENT TYPE: Conference

LANGUAGE: English

SUMMARY LANGUAGE: English

IT Major Concepts  
Molecular Genetics (Biochemistry and Molecular Biophysics)

IT Chemicals & Biochemicals  
cDNA [complementary DNA]; **potassium-channel  
interacting protein-2**: chromosomal organization,  
localization, splice variants; voltage-gated fast-inactivating  
potassium channels

L7 ANSWER 9 OF 12 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 2000:27272 CAPLUS

DOCUMENT NUMBER: 132:149426

TITLE: Caspr2, a new member of the neurexin superfamily, is  
localized at the juxtaparanodes of myelinated axons  
and associates with K<sup>+</sup> channels

AUTHOR(S): Poliak, Sebastian; Gollan, Leora; Martinez, Ricardo;  
Custer, Andrew; Einheber, Steven; Salzer, James L.;  
Trimmer, James S.; Shrager, Peter; Peles, Elior

CORPORATE SOURCE: Department of Molecular Cell Biology, The Weizmann  
Institute of Science, Rehovot, 76100, Israel

SOURCE: Neuron (1999), 24(4), 1037-1047  
CODEN: NERNET; ISSN: 0896-6273

PUBLISHER: Cell Press

DOCUMENT TYPE: Journal

LANGUAGE: English

REFERENCE COUNT: 53

REFERENCE(S): (1) Baumgartner, S; Cell 1996, V87, P1059 CAPLUS  
(2) Bekele-Arcuri, Z; Neuropharmacology 1996, V35, P851 CAPLUS  
(3) Bellen, H; Trends Neurosci 1998, V21, P444 CAPLUS  
(5) Bhat, M; Cell 1999, V96, P833 CAPLUS  
(6) Butz, S; Cell 1998, V94, P773 CAPLUS  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

ST protein Caspr2 juxtaparanode myelinated axon; **potassium channel interaction protein** Caspr2 myelinated axon; human cDNA sequence caspr2 protein

L7 ANSWER 10 OF 12 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: 1999:186364 BIOSIS

DOCUMENT NUMBER: PREV199900186364

TITLE: Molecular identification and functional characterization of

KIP1 and KIP2.

AUTHOR(S): Gong, Jianping (1); Xu, Jia (1); Bezanilla, Magdalena (1); Derin, Rachel (1); Li, Min (1)

CORPORATE SOURCE: (1) Department of Physiology and Department of Neuroscience, The Johns Hopkins University School of Medicine, 725 North Wolfe Street, Baltimore, MD, 21205 USA  
SOURCE: Biophysical Journal, (Jan., 1999) Vol. 76, No. 1 PART 2, pp. A346.

Meeting Info.: Forty-third Annual Meeting of the Biophysical Society Baltimore, Maryland, USA February 13-17, 1999

ISSN: 0006-3495.

DOCUMENT TYPE: Conference

LANGUAGE: English

IT Major Concepts

Biochemistry and Molecular Biophysics

IT Chemicals & Biochemicals

potassium channel; KIP1: **potassium channel**

**interacting protein**; KIP2: **potassium**

**channel interacting protein**

L7 ANSWER 11 OF 12 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 1997:259948 CAPLUS

DOCUMENT NUMBER: 126:340102

TITLE: Cytoplasmic domains of voltage-sensitive K<sup>+</sup> channels involved in mediating protein-protein interactions

AUTHOR(S): Scannevin, Robert H.; Trimmer, James S.

CORPORATE SOURCE: Cell Biology Inst. Cell Developmental Biology, State Univ. New York, Stony Brook, NY, 11794-5215, USA

SOURCE: Biochem. Biophys. Res. Commun. (1997), 232(3), 585-589

CODEN: BBRCA9; ISSN: 0006-291X

PUBLISHER: Academic

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

ST review voltage sensitive potassium channel domain; **protein interaction potassium channel** domain review

L7 ANSWER 12 OF 12 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 1995:79211 CAPLUS

DOCUMENT NUMBER: 122:1513

TITLE: The molecular regulation of muscarinic K<sup>+</sup> channels by GTP-binding proteins in cardiac atrial cell membrane

AUTHOR(S): Kurachi, Yoshihisa; Ito, Hiroyuki; Takikawa, Reiko; Nakajima, Toshiaki; Sugimoto, Tsuneaki

CORPORATE SOURCE: Dep. Int. Med., Mayo Clinic, Rochester, MN, 55905, USA

SOURCE: Mol. Biol. Myocard. (1992), 189-202. Editor(s): Tada,

Michihiko. Jpn. Sci. Soc. Press: Tokyo, Japan.

CODEN: 60MQAG  
DOCUMENT TYPE: Conference  
LANGUAGE: English  
IT G proteins (guanine nucleotide-binding **proteins**)  
RL: BAC (Biological activity or effector, except adverse); BIOL  
(Biological study)  
(**potassium channels** interaction with  
adenosine receptors regulation by G proteins and their subunits in  
cardiac atrial cell membrane)

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